



## Case report

Postmortem diagnosis of sudden unexpected death from *Streptococcus suis* type 2 infection: A case report

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## ABSTRACT

*Streptococcus suis* causes meningitis, septicemia, arthritis, endocarditis and death in both pigs and humans. Sudden death is rarely documented in the forensic field and almost all cases were diagnosed pre-mortem. Here we report a 49-year-old man who died from *S. suis* type 2 infection. *S. suis* was identified as the causative pathogen using bacterial culture, standard biochemical and coagglutination tests, specific DNA amplification by polymerase chain reaction, and histopathologic examination. We discuss the postmortem investigation of a suspected *S. suis* infection.

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## 1. Introduction

*Streptococcus suis* (*S. suis*) is a Gram-positive, hemolytic, facultative anaerobic coccus.<sup>1</sup> Based on its polysaccharide capsular antigens, at least 35 serotypes of *S. suis* are known.<sup>1</sup> Virulence differs among and within the various serotypes of *S. suis* and not all types cause disease.<sup>2</sup> Serotype 2 is most commonly associated with diseases in pigs and human causing meningitis, septicemia, arthritis, endocarditis and death.<sup>1,2</sup> Most of the human infection cases have been reported from Asia, mainly China and Thailand.<sup>2</sup> The probable first report of human *S. suis* infection in Thailand was reported in 1987.<sup>3</sup> The largest studies in Thailand were done in the northern part of the country, especially in the Lamphun and Chiang Mai provinces.<sup>4,5</sup> Despite numerous cases of *S. suis* infection worldwide including Thailand,<sup>6–12</sup> sudden death is rarely documented in the forensic field with most cases diagnosed pre-mortem. We report a man who died from streptococcus infection and discuss the postmortem investigation. Our case is possibly the first case report of postmortem diagnosis of *S. suis* infection in Thailand.

## 2. Case report

A 49-year-old man was found unconscious at home. His relative called the Emergency Medical Service for transport to the emergency room of the provincial hospital. The patient was apneic and pulseless. The initial cardiac rhythm was asystole. Fingertip dextrose strip examination showed 76 mg% of glucose. Cardiopulmonary resuscitation was done for thirty minutes but was not successful. Death was pronounced at 7:45 a.m. The cause of death could not be confirmed by external examinations and therefore a forensic autopsy had to be done. The son of the deceased reported that his father had been healthy without serious medical illness and allergy. The deceased consumed alcohol every evening and had a meal of raw pork from an ill pig ten days before death. The day before his death, he developed a backache without fever, jaundice, or other symptoms. The provincial hospital had no medical history of the deceased.

After death, the body was transported to keep in the refrigerator of the morgue shortly. An autopsy was performed six hours after the time of pronouncement. The body was a middle aged, fairly nourished male, 169 cm in length with short black hair. There were copious amounts of a fine frothy fluid in the nose and mouth. The body was jaundiced with multiple petechiae on the chest and abdominal wall. The upper chest wall showed multiple purpuric hemorrhages with erythematous rash. There were no wounds or injection marks on the skin. The internal examination showed no

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evidence of any vital organ injury. The brain revealed congestion with diffused multiple petechial hemorrhages in the white matter of the cerebral hemispheres. The pituitary fossa had no abnormal mass. The thyroid gland was normal in shape and size. The airways showed no edema or foreign body obstruction. Both lungs showed marked edema with some adhesions. There were no abnormal masses or pulmonary thromboembolism in the lungs. The right and the left lung weighed 950 g and 900 g, respectively. The heart had a normal shape and size, but showed more advanced decomposition than other organs. All major coronary arteries were widely patent. The left and the right ventricular free wall thicknesses were 15 mm and 5 mm, respectively. Neither valvular abnormality nor congenital anomaly was observed. The heart weighed 350 g. There was no evidence of peritonitis. The liver was enlarged and had a mild pale yellowish appearance. The spleen, small bowel, large bowel, adrenal glands, and pancreas had no significant gross pathologic abnormality. There was a cortical pallor to both kidneys which is consistent with shocked kidneys. The retroperitoneal region had no blood collection. The pelvic organs showed no significant gross lesions. There were 70 mL of a light yellow fluid in the stomach. The mucosa of the stomach showed generalized gastritis. Brain, thyroid gland, heart, lung, liver, and kidney tissues were submitted for microhistologic examination. Microscopically, the cardiac muscles showed a mild degree of myocardial fiber hypertrophy and fatty change. There was one focus of subepicardial lymphocytic infiltration and congestion among myocardial fibers with multiple foci of intramyocardial hemorrhage. The lung tissues showed a focus of non-caseating granulomatous inflammation (special stains were not performed), congestion and edema. The liver tissues showed macrovesicular fatty changes. Eighty percent of the glomerular capillaries of the kidneys contained fibrin platelet thrombi (Fig. 1). There were no significant pathological changes in the brain and thyroid gland tissues. Heart blood, liver tissue, and bile content were submitted to the hospital laboratory unit for bacterial culture and identification. The preservatives for bacterial culture were same as routine pre-mortem culture, but the collection methods were different. Blood was collected from the heart with sterile technique and then preserved in hemoculture bottle for culture at the laboratory. Bile and liver were seared and swabbed following a red-hot spatula on the surface of the organs and then the swabs were preserved in media-containing tubes for bacterial culture. All specimens were stored at  $-15^{\circ}\text{C}$  in the refrigerator and then sent for culture shortly. Viridans streptococci were

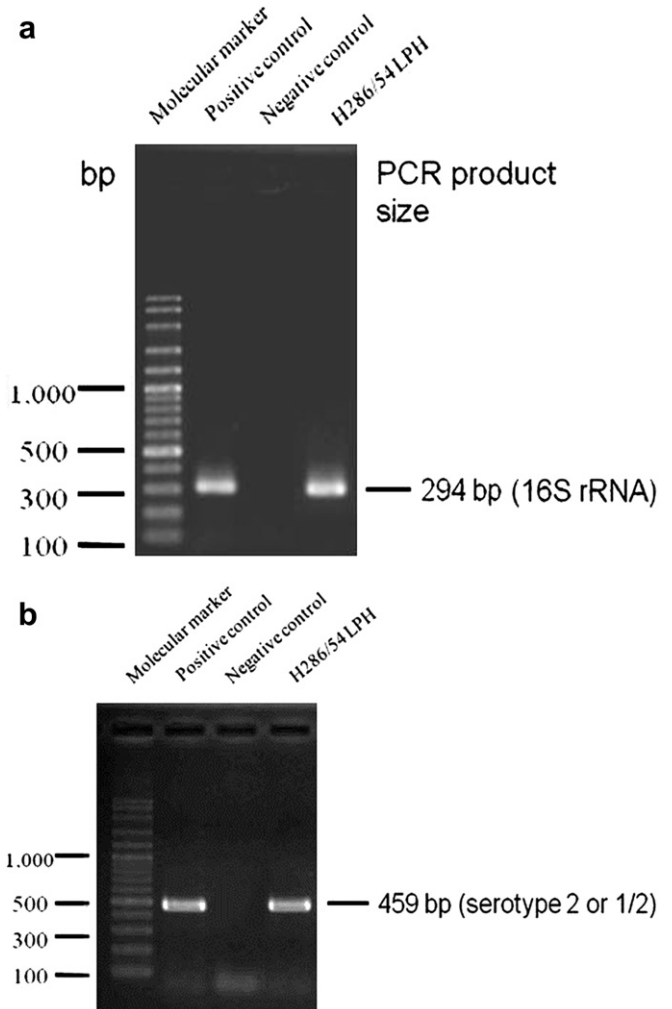
presumably isolated from all specimens. A positive hemoculture was sent to the Department of Microbiology, Faculty of Medicine, Chiang Mai University for *S. suis* identification.

### 2.1. Isolation of *S. suis* from positive hemoculture

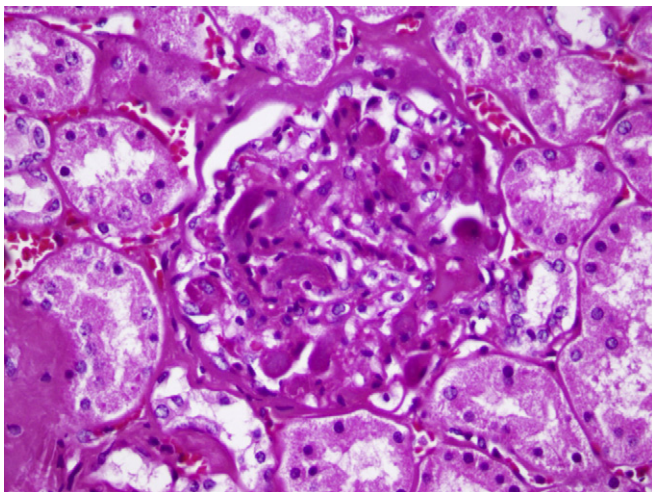
Bacteria were cultured on sheep blood agar. Alpha-hemolytic gram positive cocci were found and identified as *S. suis* by standard biochemical tests (optochin, bile esculin, mannitol and sorbitol negative; but esculin, trehalose, lactose and starch positive). In addition, the *S. suis* specific 16S rRNA gene could be amplified by PCR from these bacteria, thus confirming that *S. suis* was isolated (Fig. 2(a)). The *S. suis* serotype 2 was identified by the coagglutination test using anti-serotype 2 antibodies and detected by PCR target to *cps2* gene (Fig. 2(b)). The *S. suis* P1/7 reference strain was used for positive control.

## 3. Discussion

Human *S. suis* infections usually occur in people who are dealing with pigs or pork.<sup>2</sup> In Northern Thailand, 88.7% of the infected cases consumed raw pig meat or blood before they fell ill.<sup>11</sup> Based on information from the deceased's son, the assumed incubation



**Fig. 2.** Identification of *S. suis* in the hemoculture of the deceased. DNA was extracted from the streptococcal-positive hemoculture of the deceased (H286/54 LPH) and amplified with primers specific for (a) *S. suis* 16S rRNA and (b) the *S. suis* serotype 2 *cps2* gene.



**Fig. 1.** Photomicrograph of the kidney tissue of the deceased (40 $\times$  magnification, H&E staining). There are numerous fibrin platelet thrombi within the glomerular capillaries.

period of this case was 9 days and he died next day after developed only a nonspecific symptom (backache). Although the incubation period of severe cases was varying from study to study, it was mostly one or two days. Two studies which had small sample size showed that incubation period was no longer than one or two days.<sup>11,12</sup> Another study showed that the incubation period for streptococcal toxic shock syndrome was vary from 9 h to 9 days (median 1.6 days) and fatal toxic shock cases progressed from onset to death in a median of 25 h (range 8 h–10.5 days).<sup>8</sup> Therefore, the incubation period of severe *S. suis* infection can be up to 9 days based on previous researches and this case. The medical professional should consider this infection as one of differential diagnoses for patients who eat raw pork within ten days before onset of symptoms. Additionally, severity of *S. suis* infection may be caused by virulent factor genes, superantigens, and specific strains of the organism.<sup>11</sup> These factors may play an important role in causing different incubation period. One possible explanation for the delay of onset of symptoms might be that the deceased may have consumed only a small amount of raw pork that caused gradual bacteremia. The deceased developed symptoms gradually without any treatment and he was drinking alcohol every day. Consequently, untreated infection led to his death. Human *S. suis* infections are most frequently manifested as purulent meningitis, septicemia or septic shock.<sup>2</sup> *S. suis* is sensitive to antibiotics, including penicillin, ceftriaxone, cephalosporin, ampicillin, and amoxicillin.<sup>2</sup> The mortality rate in all clinical presentations is approximately 28%,<sup>11</sup> but 80% or more of the septic shock cases usually die.<sup>2,11</sup> All of the death cases were diagnosed by pre-mortem examination, postmortem diagnosis of *S. suis* infection is more difficult. The only way to diagnose *S. suis* is perform bacterial culture from blood (hemoculture) and tissue. However, careful history taking from relatives, external examination on the body, and high index of suspicious for serious infection may help for diagnosis and investigation. Multiple petechial hemorrhages on the non-dependent parts of the chest and abdomen with multiple purpuric subcutaneous hemorrhages on the skin of the deceased suggested that he may die of severe infection; therefore the useful additional investigations were histological examination and microbiology which revealed pathologic changes and pathogen related to the cause of death. A positive hemoculture for *Streptococcus viridans* should be tested to identify *S. suis* using standard biochemical and specific PCR tests since *Streptococcus pneumoniae*, *S. viridans* or group D enterococci form similar colonies.<sup>13</sup> Unfortunately not all necessary methods for the identification of *S. suis* are available in most microbiological laboratories.<sup>13</sup> Postmortem contamination should be considered for bacterial culture in dead body due to bacterial overgrowth. However, it was thought that the chance of contamination in this case was less likely because the following reasons: (1) This case died at the Emergency room, the body was kept in the refrigerator at the morgue shortly after pronounced death. (2) Body tissues of patients dying without clinically apparent infections are still sterile without bacterial and fungal overgrowth for at least 20 h after death.<sup>14</sup> (3) The postmortem blood and body tissues of this case were stored at –15 Celsius in the refrigerator and then sent for culture shortly. (4) *S. suis* was the only pathogen isolated from multiple sites (blood, liver tissue, and bile). (5) Processes of specimen collection for culture were performed with standard technique and used new sterile instruments, and (6) A postmortem contamination of *S. suis* has so far not been reported. In our case, the positive blood culture

combined with PCR identification as well as liver tissue and bile content examination indicate a disseminated pre-mortem infection and exclude a postmortem contamination. Unfortunately, no raw pork from this case was left for a confirmative isolation of *S. suis*. The micropathological results were consistent with diffuse intravascular coagulopathy (DIC) and the probable cause of death was toxic shock syndrome. DNA sequencing might reveal further details of the genotype of this particular *S. suis* strain. In conclusion, the skin manifestations, micropathological results which were consistent with DIC, and specifically identified postmortem microbiology were sufficient to diagnose toxic shock syndrome due to *S. suis* infection. The forensic pathologists who deal with sudden death cases should not overlook a history of raw pork consumption, especially in endemic areas. A carefully performed autopsy can readily diagnose the cause of death from *S. suis* infection. Besides, this case is a lesson for people who like to eat raw pork.

#### Ethical approval

Not applicable.

#### Funding

No funding was received.

#### Conflict of interest

This research complies with the current laws of Thai and has no conflict of interest.

#### Appendix A. Supplementary material

Supplementary data related to this article can be found online at <http://dx.doi.org/10.1016/j.jflm.2012.09.002>.

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